A CASE OF GLIOMATOSIS OF THE SPINAL CORD (AND SYRINGOMYELIA), WITH RECURRENT HÆMORRHAGES.

BY CHARLES L. DANA, M.D.

IN reporting the following case, it affords an opportunity of calling attention to the, at times, awkwardness and inapplicability of the word "syringomyelia," and of suggesting that the term "gliomatosis" of the cord would be a better generic name. In a good many cases, the cavity of the spinal cord is a small and secondary matter, having little to do with the production of the symptoms. In all cases, the primary and essential thing is the gliomatous change and infiltration produced by the new growth. There are cases of gliomatosis with very little cavity formation, my own being an example of this kind. There are, on the other hand, often cavities in the spinal cord with which there is no gliomatosis and with which few symptoms occur. In hydromyelia, for example, it is well known that the cord may have a cavity and but slight evidence of it be present. I have reported a case in which there was a number of small cavities in the anterior horns of the spinal cord produced by hæmorrhages and associated with multiple neuritis. The main symptoms were caused, undoubtedly, by the neuritis, and the syringomyelia was of little practical importance.

I do not know of any other disease which is named after the hole in it.

The study of gliomatosis or gliomatous infiltration of the nervous centres, might well be taken up from an independent standpoint and discussed just as we discuss syphilis or tuberculosis of the nerve centres. The result of abandoning the word "syringomyelia," and substituting for it "gliomatosis," with or without cavity formation, would be, I believe, an advantage to our nosology, and a help in our clinical and pathological work.

The following case was one of a gliomatous infiltration of the spinal cord in which the symptoms simulated very much a transverse myelitis. The disease progressed very slowly, and the patient died finally from a severe intramedullary hæmorrhage. The autopsy showed that while there was a slight amount of cavity formation, yet the gliomatous infiltration with secondary hæmorrhages was the distinguishing pathological feature. In the clinical history and the pathological changes there are instructive features:

J. K., *æt.* 35, German, was admitted to my service in Bellevue Hospital, October 15, 1891. He had been for some time under the care of Dr. T. J. Kearney, of this city, and I am indebted to him for the opportunity of studying the case.

The history given of his symptoms was that he had been a healthy man, married, and had been a hard-working laborer, never infected with syphilis, and moderate in alcoholic indulgence. About fifteen months before admission, he noticed first of all a pain in the right leg extending down along the course of the crural nerve, or, as he described it, running from the right groin through to the back. The pain was dull in character, but sometimes sharp, and was, he said, worse after eating. The leg felt numb and heavy and after a time it became weak and stiff, especially if he sat for a time. The pain and numbness extended slowly into the left leg. After suffering a short time with these pains, he suddenly lost power in both legs and for three months was confined in bed with paraplegia. He gradually improved and he got so that he could walk about, though with considerable difficulty. In this state of partial paraplegia he was admitted to the hospital, with the suggestion from Dr. Kearney that possibly he had a tumor of the spinal cord.

Physical examination at the time of admission showed him to be a person of medium height, well nourished and muscular. Thoracic and abdominal viscera normal. He complained of pain in the right groin and back, as described above. There was great exaggeration of the reflexes of the legs, both deep and superficial. The legs were very weak and stiff, but the right one was much more seriously involved There was anæsthesia which was chiefly marked in the right leg and extended up to the level of the twelfth dorsal spine. There was no atrophy, no especial ataxia, some weakness of the bladder and constipation, and occasionally a loss of sensation during urination. There was some involuntary twitching, chiefly of the muscles of the right leg. The patient presented the general clinical picture of a case of transverse myelitis. The possibility of his having a spinal tumor was considered, but I did not feel justified in saying that such tumor existed. The tests for anæsthesia were made on several occasions and the records show that this was complete for all forms of sensibility. There was no disturbance of the arms or face. The pupils were normal and there was no visual trouble. The patient remained in about the same condition for two months and then passed out from my observation. Nine months afterward, and about two and a half years from the time of onset of his trouble, he was readmitted to the hospital. At that time there was a total paralysis of motion and sensation in both legs. The reflexes were greatly exaggerated. He was unable to pass his water and it had to be drawn with the catheter. The bowels were constipated. The anæsthesia reached as high as the twelfth dorsal spine. He suffered the most intense paroxysms of pain which were felt across the abdomen. He also had severe headaches. He slept very little on account of the intense pain, and chloral and morphine seemed to have but little effect in deadening it. He had on the second day some involuntary fecal evacuations and occasionally he vomited. He grew rapidly weaker and on the fourth day fell into a stupor, aand died from exhaustion. He had during the third day of his stay a temperature of 101°; the rest of the time his temperature was normal.

The post-mortem showed nothing particularly abnormal except as regards the spinal cord. On opening the spinal canal, the meninges were seen to be perfectly normal; there was no congestion or discoloration or thickening and no exudate. There was a fusiform enlargement of the spinal cord at the level of the eighth dorsal segment and extending up and down for about two and a half inches. The cord was swelled to twice its normal size, but showed absolutely no external change. On making a section it was found that in the middle portion of the swelling there was a solid clot of comparatively fresh character occupying the centre of the spinal cord, and destroying nearly all of the tissue, leaving only a thin margin at the periphery. The hæmorrhage extended up and down for a distance corresponding with the length of the swelling, that is, somewhat over two inches, but it was less in amount at the upper and lower parts. In the neighborhood of the clot the tissue seemed abnormal, but not particularly softened. Sections above and below the lesion showed evidences of ascending and descending degeneration. The cord was hardened carefully in Müller's fluid and alcohol and subsequently stained with carmine, hæmatoxylon and Weigert's solution. Microscopical examination made by myself, assisted also by Drs. Judson C. Smith and George R. Elliott, showed that in the region of the fusiform swelling there was a considerable amount of gliomatous tissue which had taken the place of the normal cord structure and that in this gliomatous tissue were small hæmorrhages. The greater part, however, of the swelling was taken up by the fresh hæmorrhage. Fig. 8 D. The conclusion from our observation of the diseased part was, that the patient had had a centralglioma of the cord, beginning in the neighborhood of the ninth or tenth dorsal segment, and that there had been a hæmorrhage in this part in the early stage of his disease, this hæmorrhage causing the acute paraplegia from which he had suffered. He had partially recovered from this, but the process continued, and finally another much larger hæmorrhage had occurred, producing the total paraplegia and intense pains from which he finally suffered and died.

In the lower part of the diseased portion of the cord was a small focus of gliomatous tissue situated in the posterior column of the right side and lying close against the posterior horn, very much in the situation of the part which is affected in locomotor ataxia (see figure 12D). The lesion at this point I consider to have been the cause of the intense pain which he suffered for so long in the right groin and right hip. A clinical feature of interest in his case was the very great pain which he suffered during the whole course of the disease and the intense pain which accompanied the terminal hæmorrhage. It is usually stated that hæmorrhages into the substance of the cord are not painful, but in this case the reverse was true to an extraordinary extent. No doubt, the great destruction and tension put upon the cord tissues were the cause of the symptoms in this instance. The very striking and well-localized focus of gliomatous tissue in the neighborhood of the posterior horn suggests the probability that it is along here that nerves pass which are particularly affected in neuralgic disturbances due to lesions of the spinal cord. I have already expressed the opinion, however, that an attempt to find the so-called pain tracts is futile. It seems to me, probable, in the light of modern psychology, that pain is not to be classed among the special sensations, but is to be considered rather a modification of ordinary forms of sensibility and that, therefore, the attempt to find pain tracts is useless. There are painful sensations, but there are, strictly speaking, no pain nerves.

A study of the secondary degenerations of the spinal cord in this case is of some interest. The cuts which accompany this article show the course of the ascending and descending degenerated tracts. They are drawn from specimens with the help of the Edinger apparatus and represent accurately the situation of the degenerated tracts. It will be seen that although the lesion is most marked at the height of the eighth and ninth dorsal segments and that the cord is nearly cut across there, yet there is hardly any ascending degeneration of the cerebellar tracts; on the other hand, the antero-lateral ascending tracts of Gowers show distinct evidence of degeneration, (see Figs. 1 C to 5 D.)

The situation and the extent of the ascending tracts seems to differ very considerably, as shown in the various cases that have been reported of ascending degeneration. When showing this case before the New York Neurological Society, I collected a number of cases from other authors showing this variation.

Finally, with regard to the relation of hæmatomyelia to syringomyelia, the literature of the subject shows that hæmorrhages into the spinal cord complicating syringomyelia are not particularly rare, and it seems to me that it is not unlikely that a good many, if not all cases of so-called idiopathic hæmatomyelia, may be cases of syringomyelia. The increasing number of cases also of syringomyelia which present the symptoms to a great

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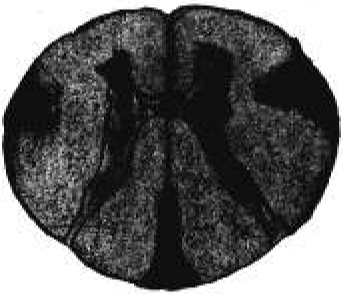
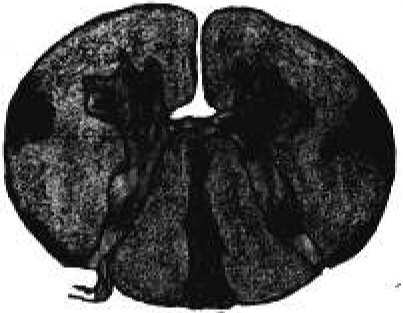


1 C



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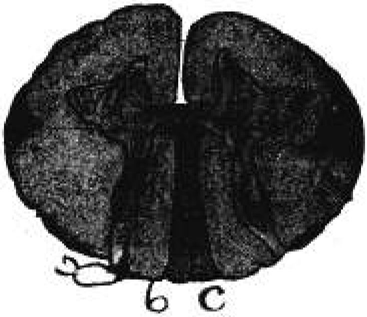

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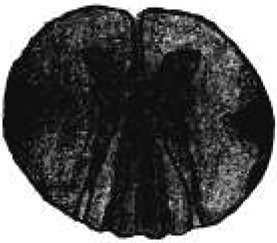

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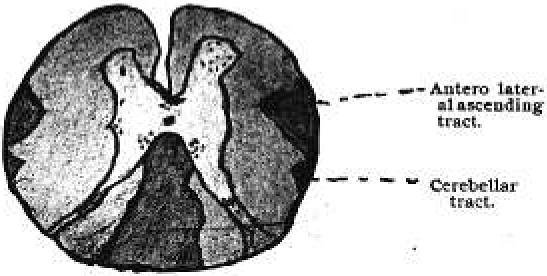

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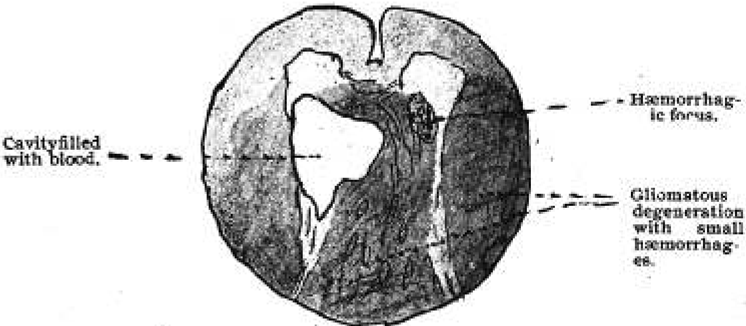
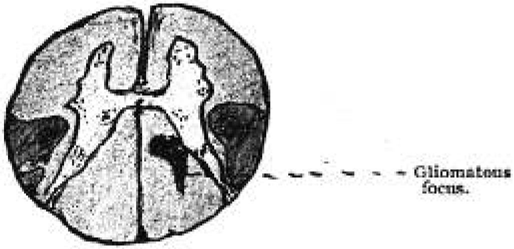
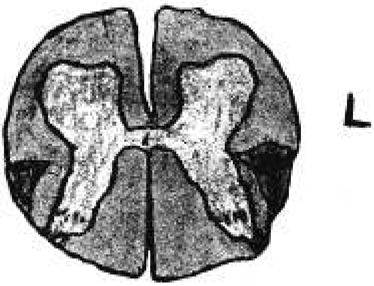


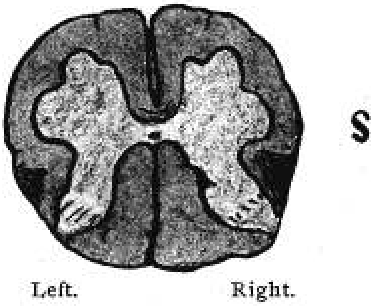
8 D



12 D

extent of transverse myelitis, leads one to think that, perhaps, there are a good many unreported cases of syringomyelia which go down in our records as cases of transverse myelitis.





SUMMARY.

The abandonment of the word syringomyelia as a clinical term and the use of the word gliomatosis or gliosis. This is recommended because there is so often gliosis without cavity.

The occurrence of spontaneous hemorrhage into the spinal cord an evidence of gliomatus in filtration; primary hæmatomyelia often an indication of some small gliomatous deposit.

The simulation by syringomyelia of acute or chronic transveres myelitis. The differentiation of disturbances in sensibility is an indication of disorganization of the central gray matter from various causes, it is not always present in syringomelia and is often present in traumatic myelitis involving the central gray.

There are probably no specific pain tracts, though there may be paths along which manifest modifications of cutaneous or tissue sensibility especially tend to travel.

Treatment of Cerebral Tumors.—At the recent eleventh meeting of the International Congress of Medicine, held in Rome, Lavista, of Mexico, spoke of the removal of tumors of the cerebrum. One of the most curious cases that has come under his observation was that of a young man who, after an extirpation three times repeated of a voluminous fibroma of the shoulder, manifested symptoms of acute encephalitis, complicated with epileptiform phenomena and paralysis of the motor oculi of the right side; coma soon followed, Suspecting the existence of a cerebral tumor situated in the corona radiata and invading the internal capsule, he proceeded to trephine, when a neoplasm presented itself, about the size of a hen's egg and having its seat in the region mentioned; this was at once removed without serious loss of blood, and great temporary relief followed. The patient subsequently died of cancer of the stomach. Lavista remarks that he knows of hardly another case of recovery after the ablation of cerebral tumors properly so called. Recovery readily takes place after removal of cysts, unless there be secondary lesions, or unless complications supervene. By itself, trephining has never contributed to aggravate the situation of patients affected with cerebral tumors; on the contrary, it has always been followed by temporary amelioration, especially in cases of superficial neoplasms. Unfortunately, in deep tumors, the disintegration of the cerebral substance renders impossible the recovery of the patient, but the surgeon should interfere all the same if he hopes to procure for the patient any benefit whatever.

In general, in encephalic lesions it will not do to wait the development of secondary complications, and the physician should operate early on patients affected with a cerebral neoplasm, for when secondary lesions have set in, it will be too late. In doubtful cases, one should first resort to an exploratory craniotomy. E. P. H.